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# Prognostic significance of electrodiagnostic studies in the Guillain-Barré syndrome[1]

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SYNOPSIS Nerve conduction and needle electromyographic studies were carried out in 50 patients with the Landry–Guillain–Barré syndrome to assess the reliability of this procedure in predicting the prognosis. Two main groups were identified. The first group was characterized by gross abnormalities in nerve conduction and absence of fibrillation potentials during the entire course of the illness. Twenty-five of 31 patients (80.6%) belonging to this group recovered rapidly, and the quality of recovery was good. In 19 patients belonging to the second group, profuse fibrillations were noted within the first four weeks of the illness with or without associated nerve conduction deficits. Recovery in this group was poor (31.5%) and pronounced residual deficits were more common. Electrophysiological studies therefore are of value not only in the diagnosis but also as a reliable prognostic index in this syndrome.

In patients with the Landry–Guillain–Barré (LGB) syndrome, the following criteria have been said to indicate a poor or incomplete recovery: cerebrospinal fluid (CSF) pleocytosis (Kaeser, 1964), papilloedema (Morley and Reynolds, 1966), profound sensory loss, or progression for more than a few weeks (Osler and Sidell, 1960). Others, however, have found these criteria to be unreliable as prognostic indices (Duvoisin, 1960; Pleasure *et al.*, 1968). Pleasure *et al.*, however, noted a positive correlation between severe quadriparesis during the acute illness and disabling residual deficits. Nerve conduction and needle electromyographic (EMG) studies were performed in 10 and nine patients respectively by these workers and a fairly good correlation was found between the severity of the residual signs and the electrical abnormalities. The results of a study of serial nerve conduction and needle electromyography in 50 cases of LGB are presented and its value as a prognostic index is discussed in this communication.

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## METHODS

Fifty patients, who were between the ages of 3 to 70 years with the LGB syndrome and were admitted to the Neurology Service of the Christian Medical College Hospital, Vellore, between 1968 and 1972, formed the basis of this study. The diagnostic criteria that we adopted were essentially similar to those described by Macfarland and Heller (1966) and Masucci and Kurtzke (1971).

Laboratory investigations, apart from routine CSF analysis, included ANF and LE cells preparations, serum and CSF specimens on admission and 14 days later for evidence of any coxsackie, ECHO, adenovirus, or polio virus infections.

ELECTRODIAGNOSTIC STUDIES Serial nerve conduction and needle electromyographic (EMG) studies were carried out in all patients, using techniques similar to those reported earlier from our laboratory (Sachdev *et al.*, 1971; Taori *et al.*, 1971). Motor conduction velocities in the median and lateral popliteal nerves were determined in all. The ulnar nerve was also studied in 39 patients. The latency and amplitude of the sensory potential evoked in the median nerve at the wrist by stimulating the digital nerves of the index finger were studied in 39 cases. Conduction velocity in the distal sensory division of the radial nerve was determined in 28 cases by antidromic stimulation in the forearm as described by Downie

TABLE 1

GROUPING OF PATIENTS ACCORDING TO NERVE CONDUCTION AND NEEDLE EMG ABNORMALITIES

| Group | | Cases (no.) |
|---|---|---|
| I | Normal nerve conduction velocities. EMG: no fibrillation. Decreased recruitment | 6 |
| II | Abnormal nerve conduction velocities. EMG: decreased interference pattern. Sporadic fibrillation potentials | 25 |
| III | Normal nerve conduction velocities. EMG: decreased interference pattern. Profuse fibrillation potentials. Polyphasic units present | 3 |
| IV(a) | Abnormal motor and sensory nerve conduction velocities. EMG: decreased interference pattern. Profuse fibrillation potentials. Polyphasia and positive sharp waves present | 9 |
| IV(b) | Abnormal motor conduction. Normal sensory conduction. EMG: decreased interference pattern. Profuse fibrillation potentials. Polyphasia and positive sharp waves | 7 |

and Scott (1967) and in the sural nerve in 35 cases. Electromyographic activity using concentric needle electrodes was sampled from the proximal and distal muscles of the upper and lower limbs bilaterally. These studies were carried out on a Medelec model MS3R Electromyograph installed in a shielded, air-conditioned laboratory with a constant temperature of 27.8–30°C.

All patients were reevaluated at intervals of two weeks after the first electrodiagnostic study up to the time of discharge.

## RESULTS

On the basis of the nerve conduction and needle EMG observations, the patients were divided into four main groups as shown in Table 1.

The severity of muscle weakness and incidence of muscle wasting is shown in Table 2. Table 3 shows the mean and range of CSF protein elevation in the various groups. The mean motor and sensory conduction velocities in the different groups are shown in Tables 4 and 5 respectively. In brief, the abnormalities noted were:

1. No response to nerve stimulation on recording from the appropriate muscle.
2. Diffuse slowing of conduction with prolonged distal latency.
3. Delayed conduction in the elbow–wrist and/or knee–ankle segments with normal distal latency.
4. Rarely, grossly abnormal distal latency with normal elbow–wrist and/or knee–ankle conduction.
5. Abnormalities of sensory conduction in the form of diminished conduction velocity, diminished amplitude of the sensory evoked potential, or total absence of the response on stimulation were noted either singly, or in combination, in groups II and IV(a) patients. Sensory conduction was normal in

TABLE 2

SEVERITY OF WEAKNESS AND INCIDENCE OF MUSCLE WASTING IN LOWER LIMBS

| Group | Total no. of cases | Muscle power grades (MRC) | | | | | | | | | | | | Wasting detected (no. of cases) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Proximal | | | | | | Distal | | | | | | |
| | | 0 | 1 | 2 | 3 | 4 | 5 | 0 | 1 | 2 | 3 | 4 | 5 | |
| I | 6 | 3 | 0 | 0 | 2 | 1 | 0 | 2 | 0 | 2 | 0 | 2 | 0 | Nil |
| II | 25 | 7 | 0 | 4 | 7 | 6 | 1 | 3 | 4 | 5 | 4 | 8 | 1 | 3 |
| III | 3 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 3 |
| IV(a) | 9 | 3 | 0 | 0 | 1 | 5 | 0 | 7 | 0 | 1 | 1 | 0 | 0 | 7 |
| IV(b) | 7 | 4 | 1 | 0 | 1 | 0 | 1 | 2 | 1 | 2 | 1 | 1 | 0 | 7 |

TABLE 3

MEAN CEREBROSPINAL FLUID PROTEIN VALUES IN DIFFERENT GROUPS

| Group | Mean CSF protein value (g/l) | Range (g/l) |
|---|---|---|
| I | 0.73 ± 0.25 | 0.40–1.20 |
| II | 1.14 ± 1.00 | 0.20–4.00 |
| III | 0.49 ± 0.27 | 0.25–0.85 |
| IV(a) | 2.29 ± 2.22 | 0.30–7.00 |
| IV(b) | 1.37 ± 0.63 | 0.75–2.70 |

TABLE 4

MEAN DISTAL LATENCIES AND MOTOR CONDUCTION VELOCITIES IN VARIOUS GROUPS

| *Groups* | *Median nerve* | | *Ulnar nerve* | | *Lat. popliteal nerve* | |
|---|---|---|---|---|---|---|
| | *Distal latency (ms)* | *Conduction velocity (m/s)* | *Distal latency (ms)* | *Conduction velocity (m/s)* | *Distal latency (ms)* | *Conduction velocity (m/s)* |
| Normal | 2.91 ± 0.44 | 58.84 ± 5.34 | 2.22 ± 0.21 | 57.13 ± 4.73 | 3.86 ± 0.87 | 47.76 ± 5.97 |
| I | NS<br>3.03 ± 0.58 | NS<br>54.7 ± 3.68 | NS<br>2.6 ± 0.28 | NS<br>56.0 ± 3.56 | 4.52 ± 1.31 | 50.5 ± 1.61 |
| II | *<br>6.44 ± 3.89 | *<br>42.11 ± 12.12 | *<br>4.44 ± 2.12 | *<br>42.59 ± 11.53 | *<br>6.9 ± 3.61 | *<br>35.22 ± 9.36 |
| III | 3.37 ± 0.19 | 54.5 ± 6.95 | 2.6 | 60.0 | 3.8 ± 0.1 | 46.5 |
| IV(a) | *<br>8.38 ± 7.46 | *<br>41.50 ± 9.83 | *<br>9.4 ± 6.83 | *<br>37.5 ± 9.50 | *<br>9.68 ± 7.73 | *<br>35.9 ± 5.30 |
| IV(b) | NS<br>3.18 ± 0.47 | NS<br>54.92 ± 5.50 | NS<br>2.85 ± 0.66 | —<br>61.33 ± 8.26 | *<br>4.66 ± 1.07 | *<br>33.8 ± 5.59 |

Test of significance of the difference in means between normals and other groups: NS: not significant. * Significant $P < 0.001$.

TABLE 5

SENSORY ABNORMALITIES IN LGB SYNDROME

| *Groups* | *Median nerve* | | *Radial nerve (c.v. m/s)* | *Sural nerve (c.v. m/s)* | *Other observations* |
|---|---|---|---|---|---|
| | *Latency (ms)* | *Amplitude (μV)* | | | |
| Normal | 2.01 ± 0.25 | 39.58 ± 9.67 | 62.59 ± 6.91 | 47.38 ± 5.25 | — |
| I | NS<br>2.30 ± 0.37 | NS<br>31.67 ± 13.12 | NS<br>59.33 ± 4.50 | NS<br>46.00 ± 1.50 | — |
| II | <br>2.06 ± 0.28 | *<br>19.29 ± 16.57 | *<br>46.20 ± 7.25 | *<br>42.56 ± 6.38 | Median nerve: no response in 13 patients.<br>Amplitude less than 10 μV in 5 patients<br>Radial nerve: no response in 2 patients<br>Sural nerve: no response in 8 patients |
| III | — | — | 52.5 | 48.5 | |
| IV(a) | <br>1.93 ± 0.45 | <br>30.00 ± 14.14 | *<br>53.50 ± 1.58 | *<br>41.50 ± 8.15 | Median nerve: no response in 6 patients<br>Radial nerve: no response in 2 patients<br>Sural nerve: no response in 3 patients |
| IV(b) | NS<br>2.09 ± 0.43 | NS<br>45.71 ± 25.55 | NS<br>60.50 ± 4.34 | <br>48.33 ± 4.51 | |

Tests of significance of the difference in means between normal subjects and other groups: NS: not significant. * Significant $P < 0.01$.

patients belonging to groups I, III, and IV(b).

Though involvement of the lower limbs was often more severe, in a few instances equally severe involvement of the upper extremities was also noted. The severity of weakness and the incidence of muscle wasting in the lower limbs is shown in Table 2. It is seen that wasting was commonly noticed in cases belonging to groups III, IV(a), and IV(b) (17 out of 19 cases), whereas this was noted only rarely in those belonging to groups I and II (three out of 31 cases). It is also evident that there was no correlation between the severity of the weakness

TABLE 6

MEAN INTERVAL BETWEEN ONSET OF ILLNESS AND FIRST EMG STUDY, AVERAGE PERIOD OF HOSPITALIZATION, AND PROGNOSIS IN DIFFERENT GROUPS

| Group | Total no. of cases | Mean interval between onset and maximum deficit (d) | Mean time lag between onset of illness and first EMG study (d) | Average period of hospitalization (d) | Prognosis | | |
|---|---|---|---|---|---|---|---|
| | | | | | Improved | Static | Deteriorated |
| I | 6 | 17.4 | 15.84± 5.74 | 25 | 5 | 1 | 0 |
| II | 25 | 15.82 | 21.43±21.81 | 18 | 20 | 4 | 1 |
| III | 3* | — | 98.32±63.65 | 14.6 | 1 | 2 | 0 |
| IV(a) | 9 | 12.5 | 31.55±15.62 | 51 | 5 | 4 | 0 |
| IV(b) | 7 | 16.8 | 19.00± 7.55 | 61.1 | 0 | 7 | 0 |

* Two of these three patients were seen three and six months respectively after the onset of the disease.

and the incidence of muscle wasting. Another striking feature was the presence of profuse fibrillation potentials in cases belonging to groups III, IV(a), and IV(b), whereas this was absent or minimal in cases in groups I and II. There was a positive correlation between muscle wasting and the occurrence of profuse fibrillation potentials, the two often occurring together in cases belonging to groups III, IV(a), and IV(b). This difference could not be explained on the basis of the time lag between the onset of the disease and the first electromyographic studies in the various groups (Table 6). It is seen that the average time interval between the onset of the disease and the first electromyographic examination was essentially similar in groups II and IV(b). Moreover, follow-up studies at weekly or two weekly intervals in groups I and II did not show the development of any significant degree of fibrillation potentials. From Table 6 it is also evident that the average period of hospitalization was prolonged in cases belonging to groups IV(a) and IV(b). The recovery was slow, and poor in the majority of cases belonging to this group in comparison with patients of groups I and II. The total number of cases in group III was very small, and two of them were seen three and six months, respectively, after the onset of the disease. These patients came to us mainly because their recovery was very slow. Their average hospital stay was only 14.6 days as none of them was acutely ill and they were discharged soon after investigations and learning physiotherapy.

## DISCUSSION

Abnormalities of nerve conduction in LGB syndrome have been reported earlier (Peterman *et al.*, 1959; Bannister and Sears, 1962; Humphrey, 1964; Isch *et al.*, 1964; Lambert and Mulder, 1964; Bergamini *et al.*, 1966; Pleasure *et al.*, 1968; McQuillen, 1971). However, the time of appearance of the conduction abnormalities has been noted to be extremely variable. Peterman *et al.* (1959) found that the conduction velocity in the nerves was normal, or only slightly reduced, in patients examined during the first few weeks of the illness, but was definitely below the normal range in patients examined eight to 50 weeks after the onset of the disease. On the other hand, Isch *et al.* (1964) and Bergamini *et al.* (1966) noted gross abnormalities of conduction at the very onset or during the first few weeks of the illness. Of the 49 cases studied by Lambert and Mulder (1964) during the first three weeks of the illness, seven (14%) had no abnormality of conduction despite the typical clinical features. Thirty cases (61%) showed a decrease in conduction velocity which was less than 70% of the normal mean and 12 (25%) showed a delay in the distal latency with slight or no slowing of the conduction in the fastest fibres. In the present series, the first nerve conduction and needle EMG study was done within four weeks of the onset of the illness in all patients except the three cases belonging to group III. In nine patients (16%)—that is, six belonging to group I and three to group III—no

abnormality in nerve conduction velocity either motor or sensory was detectable. In group I cases, where the clinical and CSF findings were typical of LGB syndrome, the average time lag between the onset of the illness and the first electromyographic study was 15.8 days. However, even on subsequent examinations at weekly intervals, no abnormality in conduction was detectable in this group, suggesting that the lesion was perhaps more proximally placed in the peripheral nervous system. Similarly in group III also, where the average time interval between the onset of the illness and the first electromyographic study was 98 days, normal peripheral nerve conduction suggested a proximal lesion. Three of the six patients belonging to group I were followed up for six, 12, and 15 months respectively and at no stage was a delay in nerve conduction detectable in them. In groups II, IV(a), and IV(b), abnormalities in nerve conduction were noted at the time of the first electromyographic examination which was carried out on an average 22, 31.5, and 14 days respectively after the onset of the illness. Abnormal motor conduction was detectable as early as three to five days in three cases belonging to group II who sought medical attention early in their course of the illness. An interesting observation was that, in these groups, the degree of slowing of conduction did not correlate with the severity of muscle weakness.

Reports dealing with abnormalities of sensory conduction in LGB syndrome appear to be rather sparse in the literature (Bannister and Sears, 1962; Pleasure *et al.*, 1968). Abnormalities of sensory conduction were noted only in patients belonging to groups II and IV(a) in the present series. Of the 25 cases in group II, the sensory conduction in the median nerve was studied in 20. No evoked potential could be recorded at the wrist in 13 patients and, in the remaining patients, the amplitude of the evoked popliteal was significantly lower than in the normal subjects, being less than 10 $\mu$V in five patients. However, the latency of the response was normal in these cases.

The conduction velocities in the radial and sural nerves were also significantly delayed in this group when compared with normal subjects. No evoked potential could be recorded from the radial nerve in two patients and from the sural nerve in eight patients. In group IV(a) the sensory evoked potential in the median nerve was not recordable in six of the nine cases. In the remaining three cases, both the latency and the amplitude of the response were found to be normal. However, in these three cases and in some of the others the conduction velocity in the radial and sural nerves was significantly delayed.

Various studies have established that marked nerve conduction defects are due mainly to myelin changes (Bannister and Sears, 1962; Kaeser and Lambert, 1962). Conversely, in Wallerian degeneration only mild slowing of conduction was noted even just before total failure of transmission. Thus, in patients where a marked slowing in conduction velocities was noted (groups II and IV(a)), it is likely that the myelin of the peripheral nerves in the segments examined was affected significantly. Conversely, in the six cases in group I, no abnormality of nerve conduction was noted and needle EMG did not show any fibrillation potentials. Therefore, one could presume that in these patients there was no significant demyelination of the peripheral nerves in the segments examined. A demyelinating process involving the proximal portions of the nerves or the nerve roots is a distinct possibility.

In the patients belonging to groups III and IV(b), profuse fibrillation potentials were noted on needle EMG. In the other groups (groups I and II), fibrillations were not noted to any significant degree even on follow-up studies. Peterman *et al.* (1959) and Rodriguez and Oester (1961) noted comparatively little fibrillations in their cases. In contrast with the gross alteration in nerve conduction velocity produced by myelin damage, the fibrillation potential is the cardinal electromyographic sign of axonal or neuronal cell body damage (Rodriguez and Oester, 1961). The time interval for the appearance of the fibrillation potential after the damage to the axon depends upon factors such as distance from the point of severence to the muscle fibre affected. In general, it may take about 15 to 20 days or even less if the lesion is very close to the muscle. These authors also state that the extent of Wallerian degeneration will be reflected in the amount of denervation fibrillations found. Thus, in the present study,

it is obvious that in patients belonging to groups III, IV(a), and IV(b) there was significant axonal damage. This axonal involvement could either be due to direct damage to the axon or the neuronal cell body or be secondary to severe myelin loss. Axonal damage along with demyelination in the LGB syndrome has been demonstrated histopathologically (Haymaker and Kernohan, 1949; Asbury *et al.*, 1969). In group IV(a), where marked slowing of nerve conduction (both motor and sensory) was noted along with profuse fibrillation potentials, it is possible that the axonal damage was secondary to severe myelin loss, even though primary axonal damage and secondary myelin loss cannot be completely ruled out.

In contrast, in group IV(b) the sensory conduction in the median, radial, and sural nerves were all within normal limits. Conduction velocities in the motor fibres of the peripheral nerves in which transmission had not failed totally were often only mildly affected. Near normal or slightly delayed motor conduction velocities with normal sensory conduction in the peripheral nerves have been reported earlier with centrally placed lesions involving the anterior horn cells, such as cervical spondylosis, syringomyelia, and motor neurone disease (Gilliatt, 1961). Gutmann and Holubar (1950) noted only a 13% reduction in conduction velocity in Wallerian degeneration even just before total failure of transmission. However, these authors have emphasized that, in Wallerian degeneration following acute lesions of the peripheral nerve, sensory transmission often fails earlier than the motor.

In this group of patients, subsequent examinations at two-weekly intervals did not show any change in the pattern already mentioned—that is, normal sensory conduction, slight delay in motor conduction in the peripheral nerves in which transmission had not totally failed, and profuse fibrillation potentials. Follow-up in two patients for 1½ and two years, respectively, did not show any significant alterations from the pattern originally noted. These data therefore suggest that, in group I, the lesion was more centrally placed with regard to the peripheral nervous system and that the brunt of the disease was borne by the axons and/or the neuronal cell bodies themselves. If the degree of slowing of conduction be a reflection of the degree of demyelination, then myelin loss, at least in the peripheral nerve segments tested in these patients, would appear to be minimal, if any.

Histopathological observations in a series of 25 cases of acute lower motor neurone paralysis clinically indistinguishable from the LGB syndrome have been reported recently (Ramos-Alvarez *et al.*, 1969). In 15 of these cases, extensive chromatolysis or striking argyrophilic degenerative changes in the neurones of the anterior horn were noted, with only minimal myelin changes in the peripheral nerves. In the remaining 10 patients, myelin changes resembling those seen in the LGB syndrome were observed. Thus, from the histopathological studies of these workers and from our nerve conduction and needle electromyographic data, it would appear that, in a sizable number of cases diagnosed as LGB syndrome, the anterior horn cells may be the primary site of the disease. The cause for this primary affection of the neurones, however, remains obscure. Ramos-Alvarez *et al.* (1969) were unable to find any definite evidence of a viral infection in any of their cases. A viral aetiology could not be proved in the present study either. Evidence available at present suggests that the LGB syndrome is a cell-mediated immunological disorder predominantly affecting the myelin of the peripheral nerves. That a similar disturbance could primarily involve the motor neurone, either its cell body or the axon, is a distinct possibility.

The prognosis as judged by the rapidity and quality of recovery was far better in groups I and II when compared with groups III, IV(a), and IV(b). Twenty-five of the 31 cases in groups I and II had improved considerably at the time of their discharge between the second and third weeks after admission. Conversely, the condition of 13 of the 19 cases belonging to groups III, IV(a), and IV(b) remained static and only six had shown any improvement even at the end of 1½ to two months in hospital and the institution of active rehabilitation procedures. Muscle wasting was also pronounced in the latter groups, being noted in 17 of the 19 cases, whereas it was present in only three of the 31 cases belonging to the former groups. From the previous discussion, it would therefore appear that in groups I and II there existed a pre-

dominantly demyelinating pathology without significant axonal loss, whereas in group III, IV(a), and IV(b) there was marked axonal damage. Pleasure *et al.* (1968) noted a good correlation between the severity of residual clinical deficits and abnormalities of nerve conduction. In a series of experimental studies, Robert and Oester (1970) demonstrated the clinical differences between a physiological conduction block and surgical section of a peripheral nerve. In the former, wasting was minimal or absent. There was no spontaneous muscle activity and complete recovery was noted within a short time after restoring conduction. However, in the latter group, marked wasting of the muscles and profuse fibrillations were observed and recovery was often very poor and prolonged.

Thus, on nerve conduction and needle EMG studies and to a lesser extent on clinical grounds, two types of patient population are noted in the LGB syndrome. In the first, in which a significant degree of fibrillation potentials is singularly lacking, even on repeated examinations, and in the majority associated with marked deficits in the peripheral nerve conduction, demyelination would appear to be the main pathological change, with axonal damage, if any, being minimal. The prognosis in this group is good. In the second type, profuse fibrillation potentials, often noted within the first four weeks after the onset of the disease, indicating significant axonal damage, is the most striking feature. Associated abnormalities in conduction may or may not be present and the prognosis is uniformly poor. Parameters such as sensory impairment, CSF pleocytosis, and the degree of rise in CSF proteins are not helpful in differentiating the two types.

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